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The study showed that a reduction in serum aldosterone was observed with a reduction in serum potassium.

When introducing elements of the present invention or the preferred embodiments(s) thereof, the articles “a”, “an”, “the” and “said” are intended to mean that there are one or more of the elements. The terms “comprising”, “including” and “having” are intended to be inclusive and mean that there may be additional elements other than the listed elements.

In view of the above, it will be seen that the several objects of the invention are achieved and other advantageous results attained.

As various changes could be made in the above methods without departing from the scope of the invention, it is intended that all matter contained in the above description and shown in the accompanying figure[s] shall be interpreted as illustrative and not in a limiting sense.

What is claimed is:

1. A method of treating hyperkalemia in a chronic kidney disease patient in need thereof optionally being treated with an effective amount of a renin-angiotensin-aldosterone system (RAAS) agent, the method comprising:

administering an effective amount of a potassium-binding agent to the patient;

wherein the patient had a serum potassium level of greater than or equal to 5.5 mEq/L and an estimated glomerular filtration rate (eGFR) of from 15 to 44 mL/min/1.73 m<sup>2</sup> before treatment with the potassium-binding agent and wherein the potassium-binding agent is administered to the patient daily for more than 8 weeks;

wherein when the potassium-binding agent is a polymer, the polymer comprises a crosslinked cation exchange polymer other than a polystyrene cation exchange polymer, and

wherein the patient's serum aldosterone level is decreased after treatment with the potassium-binding agent as compared to the patient's serum aldosterone level before treatment with the potassium-binding agent.

2. The method of claim 1 further comprising observing an increase or stabilization of estimated glomerular filtration rate (eGFR) as compared to the patient's eGFR before treatment with the potassium-binding agent.

3. The method of claim 1 further comprising observing a decrease in the patient's serum creatinine level as compared to the patient's serum creatinine level before treatment with the potassium-binding agent.

4. The method of claim 1 further comprising observing an increase in the time to progression of end stage renal disease as compared to a chronic kidney disease patient optionally treated with a RAAS agent but not treated with the potassium-binding agent.

5. The method of claim 1 further comprising observing an increase in survival as compared to a chronic kidney disease patient optionally treated with a RAAS agent but not treated with the potassium-binding agent.

6. The method of claim 2 wherein the increase or stabilization of eGFR is maintained over more than 12 weeks during which the potassium-binding agent is administered to the patient daily.

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7. The method of claim 2 wherein the increase or stabilization of eGFR is maintained over more than 24 weeks during which the potassium-binding agent is administered to the patient daily.

8. The method of claim 2 wherein the increase or stabilization of eGFR is maintained over 52 weeks or more during which the potassium-binding agent is administered to the patient daily.

9. The method of claim 8 wherein the patient's eGFR is stabilized after treatment with the potassium-binding agent.

10. The method of claim 8 wherein the patient's eGFR is increased after treatment as compared to the patient's eGFR before treatment with the potassium-binding agent.

11. The method of claim 8 wherein the patient's eGFR after treatment with the potassium-binding agent increased by at least 4 mL/min/1.73 m<sup>2</sup> or more as compared to the patient's eGFR before treatment with the potassium-binding agent.

12. The method of claim 1 wherein the patient's serum potassium level is decreased after 2 days or more of treatment as compared to the patient's serum potassium level before treatment with the potassium-binding agent, and the decreased serum potassium level is maintained over the 52 weeks or more of treatment.

13. The method of claim 2 wherein the patient's eGFR is stabilized after treatment with the potassium-binding agent.

14. The method of claim 2 wherein the patient's eGFR is increased after 3 months or more of treatment as compared to the patient's eGFR before treatment with the potassium-binding agent.

15. The method of claim 14 wherein the patient's urine albumin:creatinine ratio (ACR) is stabilized after 3 months or more of treatment.

16. The method of claim 14 wherein the patient's eGFR after treatment with the potassium-binding agent increased by at least 4 mL/min/1.73 m<sup>2</sup> or more as compared to the patient's eGFR before treatment with the potassium-binding agent.

17. The method of claim 1 wherein the patient's serum potassium level is decreased after 2 days or more of treatment as compared to the patient's serum potassium level before treatment with the potassium-binding agent.

18. The method of claim 1 wherein the patient's urine albumin:creatinine ratio (ACR) is stabilized after 3 months or more of treatment.

19. The method of claim 1 wherein the potassium-binding agent is a zeolite.

20. The method of claim 1 wherein the potassium-binding agent is a zirconium silicate.

21. The method of claim 1 wherein the potassium-binding agent is a molecular sieve.

22. The method of claim 1 wherein the potassium-binding agent is a zirconium germanate.

23. The method of claim 1 wherein the potassium-binding agent comprises substantially spherical particles.

24. The method of claim 1 wherein the potassium-binding agent comprises particles having a mean diameter of less than 250 μm.

25. The method of claim 1 wherein the potassium-binding agent comprises particles having less than 4 volume percent of the particles having a diameter of less than 10 μm.

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